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**Medical Staff Conference**

# Heat Stroke

*These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California, San Francisco. Taken from transcriptions, they are prepared by Drs. David W. Martin, Jr., Assistant Professor of Medicine, and H. David Watts, Assistant Professor of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine. Requests for reprints should be sent to the Department of Medicine, University of California, San Francisco, CA 94143.*

DR. SMITH:* *Incredible as it may seem on this chilled June day in San Francisco, the topic of our first presentation is heat stroke. Dr. Clinton Lawrence will give the case summary.*

DR. LAWRENCE:† The case for today is that of a 22-year-old white man, a physical education major at the University of California, Berkeley, who was in good health until he collapsed near the finish line of the Bay to Breakers Race. That particular day was sunny but windy. The patient was said to be perspiring profusely at first and was confused. He was covered immediately with down coats and taken to the emergency room at another medical center. He apparently vomited and was incontinent of stool, but there was no seizure activity. His only memory of the event was of a sudden impact. On admission here he denied the use of amphetamines, antihistamines or other drugs. His medical history showed episodic wheezing during the spring for the past four years, but he had not taken medications or seen a physician for this condition. There was also a questionable history of black, tarry stools. The patient reported that upon exposure to extreme cold in Colorado during the skiing season a few years previously, he had had abnormally cold and blanching fingers.

Physical examination on admission here showed blood pressure of 130/70 mm of mercury, pulse rate of 150 per minute, respiratory rate of 32 per minute and temperature of 40°C (105°F) rectally. The patient was well-muscled, tachypneic and appeared confused. The skin was extremely hot and dry. The pupils were dilated but reactive, and there was no evidence of trauma. The neck was supple. On examination of the chest, diffuse inspiratory and expiratory wheezes were heard. Results of cardiac and abdominal examinations were normal. There was 3 plus guaiac-positive stool and nasogastric aspirate. The remainder of the examination showed no abnormalities except for disorientation and symmetrically decreased reflexes.

Admission laboratory data included a hematocrit of 49 percent, and a leukocyte count of 15,000 per cu mm with a normal cell differential. The prothrombin time was slightly prolonged at 12.7 seconds with a control of 11 seconds. At admission, electrolytes showed a sodium level of 147, potassium 5.1, bicarbonate 23 and chloride of 107 mEq per liter. Blood urea nitrogen (BUN) was 27 and creatinine was 2.5 mg per 100 ml. On

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urinalysis there was a positive reaction for protein and there was a positive Dipstix® reaction for heme products, but only a few red cells and granular casts were seen in the sediment. The patient was hypoxic and there was respiratory alkalosis. The serum content of muscle enzymes was slightly elevated, as were serum glutamic oxaloacetic transaminase (SGOT), lactate dehydrogenase (LDH) and creatine phosphokinase (CPK), but with normal aldolase at that time. An electrocardiogram showed a pronounced right axis and an x-ray film of the chest was clear. The patient was treated at first for heat stroke with intravenous fluids and mannitol to maintain diuresis. He was placed on a cooling blanket in the Intensive Care Unit and iced down with cool towels. There was an immediate response with a decrease in temperature and a clearing of the sensorium. Subsequently the patient did fairly well.

Several problems developed during the patient's stay in hospital. Renal impairment resolved somewhat and there was a BUN of 21 and creatinine content of 1.3 mg per 100 ml on discharge. There were 3.3 grams of proteinuria per 24 hours and normal creatinine clearance. Rhabdomyolysis developed, manifested by very tender and swollen thighs with extremely elevated serum content of muscle enzymes. The urine at first was clear but became quite dark again with a positive hemestick reaction, but no red cells were noted. A myoglobin determination is pending. Also, liver dysfunction developed, with jaundice. The liver was of normal size but tender, and the bilirubin content was 5.5 mg per 100 ml of blood, mainly direct. There was well developed evidence of disseminated intravascular coagulation but with no bleeding. Prothrombin time, partial thromboplastin time and thrombin time were elevated, with low platelet count and low fibrinogen and elevated fibrin-split products. All of these problems resolved during the patient's stay in hospital, and most of the serum enzyme levels were returning toward their normal ranges at the time of discharge. The only other problem was the wheezing noted on admission with the right axis deviation shown in an electrocardiogram. A lung scan was negative for pulmonary emboli. Creatine phosphokinase fractionation showed the source was predominately skeletal muscle, but there was a small cardiac fraction.

We have the patient with us today for those of you who might like to ask him a few questions.

DR. SMITH: *This patient will be discussed by Dr. Floyd C. Rector, Jr. Dr. Rector is Professor of Medicine and Chief of the Renal Division at Moffitt Hospital in our Department of Medicine. Dr. Rector joined us from Dallas last August. He assures me that this problem is not so rare in Texas as it is in San Francisco. Dr. Rector, would you like to ask any questions of the patient?*

DR. RECTOR:* How long had you trained for the race?

*Patient: About three months, seriously.*

DR. RECTOR: How frequently would you run in your training?

*Patient: Five days a week.*

DR. RECTOR: Had you taken any salt tablets at any time during your training period?

*Patient: No.*

DR. RECTOR: Was there anything different during the race as compared to your training period?

*Patient: The only difference would be that we ran through Golden Gate Park twice and the finish line was about 400 yards past where we had trained to finish; so when we got to the point where we thought the race would finish, there were about 400 yards remaining.*

DR. RECTOR: Did you sprint this 400 yards?

*Patient: Yes, I tried.*

DR. RECTOR: Do you remember anything in the period of time after you collapsed and before you arrived at the hospital here?

*Patient: I do remember after I had gone down several people trying to get me out of the way of all the other runners. I thought I was conscious at the emergency hospital and they said I was not, so I guess you call that hallucinations. I guess I was doing a little of that.*

DR. RECTOR: How have you done since you left the hospital here?

*Patient: I have tried to run about three or four times.*

DR. RECTOR: Have you?

*Patient: Yes, and it has been a mild jog.*

DR. RECTOR: Do you have any muscle problems now?

*Floyd C. Rector, Jr., MD, Professor of Medicine, Chief of Renal Division, Department of Medicine.

TABLE 1.—*Three Syndromes Associated with Heat*

*Heat Cramps*

Muscle cramps following exertion in hot weather due to salt depletion

*Heat Exhaustion*

1. Water depletion—Thirst, fatigue, weakness, anxiety, hyperventilation, paresthesias, agitation, psychosis, *hyperthermia*
2. Salt depletion—Muscle cramps, weakness, headaches, giddiness, anorexia, nausea, vomiting and diarrhea, hypotension, tachycardia, *body temperature normal*

*Heat Stroke*

Hyperpyrexia (rectal temperature greater than 41.1°C), anhidrosis (present in only 50 percent of cases), delirium, coma

*Patient: Just a little tenderness. I have lost a lot of weight in my legs.*

DR. RECTOR: Do you still have any discrete tender spots?

*Patient: No, not really.*

DR. RECTOR: Are there any signs of nodules developing in your legs?

*Patient: No.*

DR. RECTOR: Thank you, Mr. E.

DR. RECTOR: Mr. E. had a rather serious heat illness which was precipitated by exceptional exertion. The degree of exertion appears to be greater than he had experienced in his training period. This illness was characterized by delirium, hallucinations, diminished consciousness and finally a stuporous state, hyperpyrexia and extentensive tissue damage. He had evidence of diffuse capillary endothelial damage with secondary intravascular coagulative syndrome. He had evidence of a mild myocardial involvement, as well as some elevation of liver enzymes. There was extensive rhabdomyolysis with myoglobulinuria and a transient episode of renal insufficiency which rapidly cleared. This syndrome was called heat stroke, although it is not typical of the classic definition of heat stroke as originally defined by the British Medical Council.

Table 1 lists three syndromes associated with heat.[1] There are heat cramps, heat exhaustion and heat stroke. Heat cramps follow fairly strenuous exertion in hot weather, occur primarily in well-acclimatized, physically fit individuals, are associated with muscle cramps, are due entirely to salt depletion and can be corrected by salt replenishment. The second syndrome is heat exhaustion, of which there are two forms. One form, which is due mainly to water depletion, tends to occur in older debilitated people or young children who, for one reason or another, are unable to replenish sweat losses by drinking. They become dehydrated and hypernatremic, have intense thirst, fatigue and weakness, become irritable and develop paresthesias, agitation, hyperthermia and sometimes psychosis. The second form of heat exhaustion is related to salt depletion, which in some individuals reaches deficits as great as 1,000 mEq. The symptoms, which can be attributed largely to extreme contraction of extracellular volume, include muscle cramps, weakness, headaches and orthostatic hypotension. The body temperature tends to be normal or even subnormal. Either of these two forms of heat exhaustion may progress to heat stroke, which classically is defined as hyperpyrexia with a rectal temperature greater than 41.1°C (106°F), cessation of sweating, delirium and coma.

The illness of Mr. E. was called heat stroke, although the recorded temperature was only 40 to 40.5°C, or about 105°F. He continued to sweat immediately after the first episode of collapse but had ceased to sweat by the time he arrived at the emergency room, and he did have the typical central nervous system symptoms. The classic definition of heat stroke refers primarily to the type of heat stroke that develops in older individuals, alcoholics and people with chronic debilitating illnesses, particularly those with underlying heart disease. In these people, heat stroke tends to develop during extended periods of high environmental temperatures in which both the day and night are hot. They sweat over a period of three or four days, gradually there is exhaustion of sweating and heat stroke develops. These patients classically have anhidrosis and dry skin when first seen.

In recent years, however, another type of heat stroke associated with extreme exertion has been seen with increasing frequency. This usually occurs in young athletes, particularly football players during spring training and in the beginning of the fall football season, and young recruits in the military who undergo basic training in the Southern states during the summer months. When heat stroke develops in these persons, the rectal temperatures may not be as great as 41.1°C, yet there still is considerable tissue damage from hyperpyrexia. First, it seems that in severely exercising persons there is a pronounced

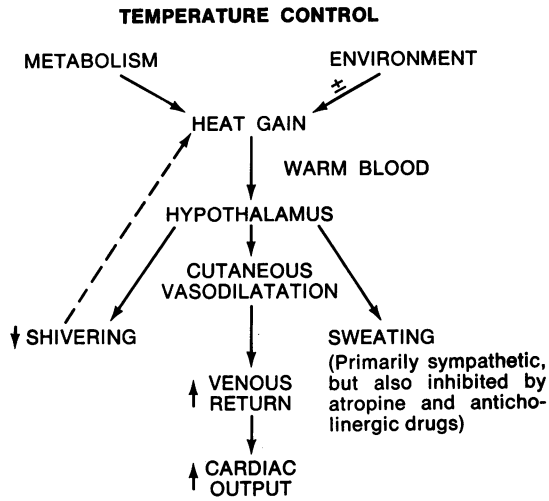


**Figure 1.**—Diagrammatic representation of temperature control in man.

temperature gradient between hepatic and rectal temperatures. In young men vigorously exercising in a hot climate Rowell et al[2] found that the temperature of the hepatic venous blood may be 106 to 107°F, while rectal temperature is only 102 to 103°. Second, when heat stroke develops in young persons after severe exertion, there is not necessarily sweat exhaustion and they may be sweating at the time the heat stroke develops. Thus, there are some differences between the classic form that occurs in older debilitated individuals or alcoholics, and the form occurring in young athletes.

As a background to considering the pathogenesis of heat stroke, it is helpful to consider a few aspects of temperature control (Figure 1). The body gains heat either from that produced by metabolism or from the environment if the environmental temperature is greater than body temperature. The blood which is warmed by this process is monitored in the anterior hypothalamus where there exists a temperature-sensitive area responsible for coordinating heat dissipation. When warm blood enters the hypothalamus, the latter will stimulate sweating. The sweating is primarily sympathetically enervated so that any sympatholytic drug may block it, but it is also inhibited by atropine and anti-cholinergic drugs. The essential purpose of sweating is to cool the skin. The second phase of heat dissipation is to increase the flow of blood to skin where it can be cooled. Thus, cutaneous vasodilation increases venous return and cardiac output so that there is a high output state. Finally there is decreased

TABLE 2.—*Causes of Heat Stroke*

| | |
|---|---|
| I. | Increased heat production |
| | A. Extreme exertion |
| | B. Agitation—delirium tremens, psychosis, drugs (LSD, amphetamines) |
| II. | Impaired heat dissipation |
| | A. Impaired sweating |
| | 1. Skin diseases, cystic fibrosis |
| | 2. Drugs—atropine, belladonna |
| | 3. Mechanical—surgical drapes, football uniforms |
| | B. Cardiovascular disease |
| | C. Central nervous system—cerebrovascular disease, alcohol, tranquilizers |

shivering which reduces heat production. These are the three components integrated at the hypothalamic level that serve to regulate body temperature.

Heat stroke may occur as the result of abnormality at one site or several different sites. There might be production of energy at a rate greater than it can be dissipated or there might be some interference with the dissipation process.

Table 2 shows some of the causes of heat stroke. Heat stroke may result from increased heat production, either from extreme exertion, as illustrated by our patient today, or from extreme agitation. This is sometimes seen in patients with alcoholic withdrawal and delirium tremens. If hyperpyrexia develops during the course of delirium tremens, it is associated with a very high mortality rate. There are certain psychotic patients who have become agitated and developed heat stroke, and finally certain drugs, particularly LSD (lysergic acid diethylamide) and amphetamines, have induced sufficient agitation and hyperactivity to precipitate heat stroke. Heat stroke also may develop from impaired heat dissipation. This can be due to impaired sweating. A variety of skin diseases involve the sweat glands and prevent sweating, or there may be congenital absence of sweat glands. Cystic fibrosis does not necessarily impair sweating but because of very high sweat, sodium chloride concentration may lead to salt depletion. Effective sweating may be prevented by either certain drugs or mechanical covering of the skin. At one time when surgical suites were not air conditioned, a number of patients developed heat stroke as a consequence of impaired heat dissipation secondary to the surgical drapes. Football players may also have impaired sweating as a result of their uniforms. Approximately 30 percent of the sweating skin is covered by nonporous

TABLE 3.—*Exertion-Induced Heat Stroke*

I. Electrolyte and acid-base disturbances
  A. Hyperventilation, with respiratory alkalosis
  B. Lactic acidosis
  C. Hypo- or hyperkalemia
  D. Hypocalcemia early; hypercalcemia later
  E. Hyperuricemia
  F. Elevation of muscle, hepatic and cardiac enzymes

II. Hematologic
  A. Leukocytosis; eosinophilia
  B. Diffuse capillary endothelial damage with hemolysis, thrombocytopenia, consumptive coagulopathy, fibrin-split products

III. Central nervous system
  A. Delirium, stupor or coma
  B. Convulsions—localized or generalized
  C. Cerebellar dysarthria and ataxia—may be chronic

IV. Cardiac
  A. Subendocardial necrosis
  B. High output failure with hypotension and tachycardia

V. Hepatic—mild to fulminant necrosis

VI. Muscle—rhabdomyolysis; calcification

VII. Kidneys—
  A. Acute renal failure—rare in non-exertional heat stroke
    1. Myoglobinuria
    2. Uric acid nephropathy
    3. Diffuse intravascular coagulation
  B. Chronic progressive interstitial nephropathy—10 percent

leather in the usual football uniform, and this may seriously impair heat dissipation. Patients with cardiovascular disease may be unable to deliver the necessary quantity of blood to the skin surface for cooling. Finally, there may be disruption of the central nervous system or the hypothalamic integration center by cerebrovascular disease, alcohol or tranquilizers. Therefore, any of the above circumstances may lead to heat stroke.

Table 3 shows some of the features that we now associate with exertion-induced heat stroke. There are a variety of electrolyte and acid base disturbances. Such patients uniformly hyperventilate and have a respiratory alkalosis. This may have very serious consequences as far as its effect on cerebral blood flow and may contribute to the altered sensorium. Lactic acidosis may develop in some patients. It is of interest that in the case of our patient today there was only respiratory alkalosis without lactic acidosis. This may have been due to the two- or three-hour delay in arriving at our emergency room. The patients may have either hypokalemia or hyperkalemia. Approximately half of the cases of young recruits or athletes that develop heat stroke have evidence of potassium depletion, and this may play an important role in the development of their hyperpyrexic state. Against this background if extensive rhabdomyolysis develops, if there is extensive hemolysis or if lactic acidosis develops with a shift of potassium out of cells, hyperkalemia may supervene, even against the background of serious potassium depletion.

One thing that has not been widely recognized is that hypocalcemia with tetany may occur early in the course of heat stroke, particularly in those persons in which the heat stroke is induced by exertion. This is primarily due to deposition of calcium in damaged muscles. There are studies showing that labeled calcium will migrate and precipitate in damaged muscle in this condition. Later hypercalcemia may develop in such patients. It is probable, although unproven, that the initial hypocalcemia induces hyperparathyroidism which then does not shut off immediately and results in a transient period of secondary hyperparathyroidism, causing rebound hypercalcemia. With Mr. E., there did not appear to be initial hypocalcemia; in fact, transient hypercalcemia was indicated. Initial calcium was about 11 mg per 100 ml. This was probably a reflection of the concentration of plasma proteins.

In patients with exertion-induced heat stroke, there may be very high rates of uric acid production and serum uric acid levels. In our patient, there was a serum uric acid of 12 mg per 100 ml. This is very common and may contribute to uric acid nephropathy. Then, of course, there are elevations of the serum enzymes, originating from muscle, liver and heart. There are considerable hematological changes. There is almost always leukocytosis with a left shift and eosinophilia. There is also evidence of diffuse capillary endothelial damage, as shown by electron microscopy. Secondary to this there may develop diffuse intravascular coagulopathy.[3] This was present in the case discussed today. It may lead to widespread bleeding diathesis. In fact, in some of the earliest cases of heat stroke reported in the 1700's, bleeding was a very prominent symptom.

There also are pronounced central nervous system disturbances, including delirium, stupor, coma, convulsions—either localized or generalized —and cerebellar symptoms (dysarthria and ataxia). In particular, the cerebellar symptoms may persist and be chronic. The heart frequently

shows evidence of subendocardial necrosis and hemorrhage, and in a few instances involving young men, there have been through-and-through myocardial infarctions without evidence of underlying coronary artery disease. At the time of heat stroke there is a high output state with a high cardiac output, low blood pressure and a tachycardia. Hypotension may be very serious in some instances. There may be mild to fulminating hepatic necrosis. Some individuals have actually died in hepatic coma. There is extensive rhabdomyolysis. Early, calcium may precipitate in the damaged muscle and later there may be evidence of muscle calcification.

Acute renal failure is very common in the exertion-induced heat stroke, but it is rare in the classic form that occurs in older persons or alcoholics.[4] It appears that the renal failure that develops is not a result of the hyperpyrexia per se, but is related more to associated muscle damage, myoglobulinuria,[5] increased uric acid production with superimposition of uric acid nephropathy and diffuse intravascular coagulation. Renal function appears usually to recover almost totally. However, there is a report from South Africa in which 40 patients from the diamond mines had heat stroke with renal failure of an exertional type; in 10 percent of those patients a chronic progressive interstitial nephritis developed, and those were the patients tending to have slightly more severe forms of acute tubular necrosis and higher serum urate concentrations.[1]

The findings listed here characterize heat stroke as it manifests itself not so much in the classic form of a debilitated person, but in a young athlete who exercises strenuously in a hot environment. In evaluating some of the factors involved in causing heat stroke in these persons, it is helpful to review briefly the physiology of heat production, dissipation and acclimatization. If an individual exercises mildly, he may produce heat at a rate of 300 kilocalories per hour, and at peak levels—for example in a 100-yard dash—it may reach 900 to 1,000 kilocalories per hour. If a person were exercising at a mild rate (300 kilocalories per hour), and were insulated so that he was neither gaining nor losing heat from the environment then the temperature would increase approximately 9°F in a single hour. If he were exercising at a maximal rate (approximately 600 kilocalories), then there would be about an 18° rise per hour. In order to prevent these extreme increases in temperature a tremendous amount of heat must be dissipated by sweating and delivery of blood to skin. Sweat rates can reach about 1.5 liters an hour in an unacclimatized person and 2.5 to 3 liters an hour in an acclimatized individual. When vaporized, 1.5 liter of sweat is capable of dissipating maximally about 675 kilocalories per hour. Thus, an unacclimatized individual could dissipate a fairly heavy heat load provided something did not interfere with sweating or evaporation. However, sweat rates tend to drop off rapidly in the unacclimatized individuals because they may become salt and volume depleted. When this occurs, body temperature starts to rise. It is important, therefore, in persons who are going to be undergoing heavy exercise in high temperatures that acclimatization has occurred.

TABLE 4.—*Physical Conditioning and Heat Acclimatization*

| | |
|---|---|
| I. | Metabolic—increased efficiency |
| | A. Increased muscle glycogen |
| | B. Increased muscle mitochondria |
| II. | Hemodynamic |
| | A. Increased plasma and extracellular volumes |
| | B. Increased maximal cardiac output |
| | Increased stroke volume |
| | Decreased peak heart rate |
| III. | Sweating |
| | Decreased sweat sodium secondary to increased aldosterone |

Table 4 shows some of the processes involved in physical conditioning and heat acclimatization. First, there is an increase in metabolic efficiency so that there is better energy utilization. In a trained person there is an increase in muscle glycogen which provides a source of energy for protracted muscle work. There is an increase in muscle mitochondria which serves to switch metabolism from the anaerobic to the oxidative phase, a more efficient process. These changes begin early in the course of physical conditioning but may take as long as two months to become complete. The second aspect of acclimatization is hemodynamic, thus augmenting the ability to increase blood flow both to muscles where heat is generated and to skin where it is dissipated. Two aspects of the hemodynamic adaptation are (1) increased plasma and extracellular volume to compensate for the vasodilatation that occurs when one exercises in a hot climate, and (2) increased myocardial efficiency presumably secondary to increased cardiac mitochondria, thereby resulting in increased maximal cardiac output, in-

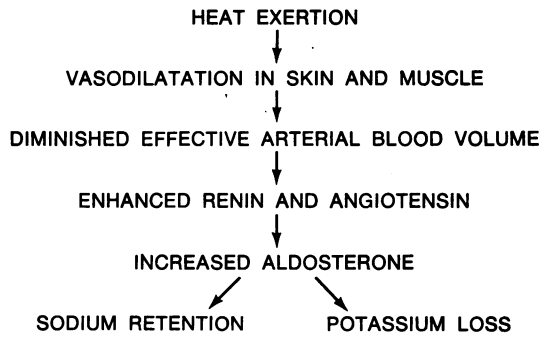


**Figure 2.**—Mechanisms leading to potassium depletion.

creased stroke volume and decreased peak heart rate during exercise. Finally, the sweating process becomes more efficient, both as far as the rate of sweating and the ability of the sweat glands to conserve sodium. This latter process is totally dependent on aldosterone. Part of this adaptive process, both as far as the increase in extracellular fluid volume and the decreased sweat sodium, depends on an adaptive increase in aldosterone.

The salutary role of aldosterone in promoting heat acclimatization has been known for a long time, but what has not been widely recognized is that the adaptive increase in aldosterone may have certain deleterious effects. This is illustrated in Figure 2. If one exercises in a hot environment there is marked vasodilation in skin and muscle which transiently decreases the effective arterial blood volume, stimulates renin and angiotensin, and an increase in aldosterone. This increase in renin, angiotensin and aldosterone occurs whether the subjects are on a low, normal or high salt diet. The aldosterone will contribute toward salt retention and expansion of the plasma volume, as well as more efficient sweating. The effect of aldosterone, however, persists after the period of exercise, and since these persons tend to be taking in salt, and in many instances very high amounts of salt, this combination of high salt intake with increased aldosterone results in some potassium loss.

In studying two groups of recruits that underwent basic training in San Antonio—one group in winter months and another group in the summer months—Knochel[6] showed that the recruits who had their physical training during the summer months lost almost 600 milliequivalents of their exchangeable body potassium. This occurred within the first week or so, and then by the fourth week the deficit had been spontaneously corrected. The mechanism for the correction is not clear.

TABLE 5.—*Consequences of Potassium Depletion*

| | |
|---|---|
| I. | Kaliopenic nephropathy with impaired urine concentration |
| II. | Decreased response to catecholamines; postural hypotension |
| III. | Metabolic<br>Decreased glycogen synthesis<br>Impaired insulin release with hyperglycemia |
| IV. | Decreased aldosterone production with an increase in sweat sodium |
| V. | Myocardial insufficiency |
| VI. | Muscle |
| | A. Increased membrane permeability with leak of muscle enzymes and decreased membrane potential |
| | B. Impaired exercise hyperemia causing rhabdomyolysis and hyperpyrexia |

The aldosterone levels increase at first and then drop off some, but there is still enough aldosterone to maintain low sweat sodium. However, for some reason the potassium deficit is replenished despite this state of mild functional hyperaldosteronemia.

Some of the consequences of this degree of potassium depletion are shown in Table 5. Kaliopenic nephropathy may develop, with impaired urine concentration and impaired water conservation. During World War II there were reports of recruits who had very high salt intakes in whom pronounced polyuria developed, with urine volumes up to 8 to 10 liters. Presumably this was a combination of kaliopenic nephropathy and high salt intakes. Potassium deficiency may also impair the response to catecholamines. As a result, there may be postural hypotension. In addition, potassium deficiency has certain metabolic consequences such as diminished glycogen synthesis, impaired insulin release with the hyperglycemic stimulus and carbohydrate intolerance. Potassium depletion may also decrease aldosterone production, which would then secondarily impair the adaptive response to the sweat glands. Knochel has recently shown that myocardial insufficiency with pulmonary edema during severe exertion may develop in dogs with potassium deficiency. Finally, there is pronounced alteration of muscle function with increased permeability of the membrane to enzymes so that there is a leakage of aldolase, CPK and other muscle enzymes in the nonexercising, potassium deficient animal. In addition, hypokalemia impairs exercise hyperemia in muscle and may lead to rhabdomyolysis and hyperpyrexia.

In Knochel's experiments, which were performed in both normal and potassium deficient dogs, the gracilis muscle was stimulated electrically, potassium release was measured in the venous effluent and blood flow to the gracilis was measured in response to stimulation.[7] Stimulation caused a pronounced increase in the release of potassium into the venous effluent in normal but not in potassium deficient dogs. In association with these changes there was a marked increase in muscle blood flow in the normal animals but not in the potassium deficient animals. This failure to increase blood flow could be corrected or partially reversed by infusion of potassium chloride into the muscle.

To summarize, persons training in a hot environment develop moderate potassium deficiency early in the course of their acclimatization. This may impair muscle release of potassium during muscle contraction, as well as cause a slight decrease in myocardial efficiency, so that together these two factors might lead to a decrease in the extent of exercise hyperemia causing muscle ischemia. As a consequence there might be rhabdomyolysis. In addition, the heat that is generated in the core of the muscle would not be effectively dissipated, leading to damage and hyperpyrexia.

Exertion-induced heat stroke is becoming an increasingly serious problem in young athletes. Unfortunately, it has a very high mortality. In certain series as many as 80 percent of the subjects who develop full blown heat stroke have died. This could be prevented by a proper period of acclimatization. There is no evidence that salt loading beyond a certain level facilitates the acclimatization. It is necessary to have a certain amount of salt supplementation, but doubling the quantity of salt into the diet is perfectly adequate. Young athletes, particularly on high school football teams, frequently are deprived of water with the misconception that this will accelerate the acclimatization process. However, *this practice is very hazardous*. It is important that young athletes go through a proper acclimatization period, that they have salt replacement, that they have adequate water intake and that they gradually work up to their full exertional activities. When heat stroke does develop, it is important that cooling be initiated as rapidly as possible by ice packs and evaporative procedures of one type or another. It is important that an airway and an intravenous needle be in place before the patients are placed in an ice bath; the procedures may be difficult to do later when they begin to convulse or vomit. There is no convincing evidence that hypothermic drugs or steroids have a beneficial effect.

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